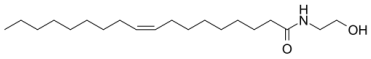
Data Sheet

Product Name:	Oleoylethanolamide
Cat. No.:	CS-0028847
CAS No.:	111-58-0
Molecular Formula:	C20H39NO2
Molecular Weight:	325.53
Target:	Endogenous Metabolite; PPAR
Pathway:	Cell Cycle/DNA Damage; Metabolic Enzyme/Protease
Solubility:	H2O : < 0.1 mg/mL (insoluble); DMSO : 20.83 mg/mL (63.99 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Oleoylethanolamide is a high affinity endogenous **PPAR-α** agonist, which plays an important role in the treatment of obesity and arteriosclerosis. IC50 & Target: PPAR-α[1] **In Vitro:** Oleoylethanolamide (OEA), an endogenous PPAR-α ligand, attenuates liver fibrosis targeting hepatic stellate cells. Oleoylethanolamide suppresses TGF-β1 induced hepatic stellate cells (HSCs) activation in vitro via PPAR-α. To assess the impact of Oleoylethanolamide on HSCs activation, the expression levels of α-SMA and Col1a in TGF-β1-stimulated HSCs are examined by qPCR. The mRNA levels of α-SMA and Col1a are markedly induced in the group of CFSC cells with TGF-β1 (5 ng/mL) stimulation for 48h, while the mRNA levels are suppressed when treated with Oleoylethanolamide in a dose-dependent manner. Immunofluorescence and western blot results show that Oleoylethanolamide treatment dose-dependently inhibits the protein expression of α-SMA, the marker of HSC activation. The inhibitory effects of Oleoylethanolamide on HSCs activation are completely blocked by PPAR-α antagonist MK886 (10 μM). Moreover, the mRNA and protein expression levels of PPAR-α are down-regulated with TGF-β1 stimulation, while Oleoylethanolamide treatment restores these changes in dose-dependent manner. In addition, the phosphorylation of Smad 2/3 is upregulated in the presence of TGF-β1 stimulation, consistent with the observed effects on HSC activation, while Oleoylethanolamide (10 μM) reduces the phosphorylation of Smad2/3 in CFSC simulated with TGF-β1[1]. **In Vivo:** Oleoylethanolamide (OEA) can significantly suppress the pro-fibrotic cytokine TGF-β1 negatively regulate genes in the TGF-β1 signaling pathway (α-SMA, collagen 1a, and collagen 3a) in mice models of hepatic fibrosis. Treatment with Oleoylethanolamide (5 mg/kg/day, intraperitoneal injection, i.p.) significantly attenuates the progress of liver fibrosis in both two experimental animal models by blocking the activation of hepatic stellate cells (HSCs)[1].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [1]**CFSC, HSC cell lines** are first obtained from cirrhotic rat liver, and have a similar phenotype to that of early passage primary HSCs. CFSC cells are cultured in Dulbecco's modified Eagle's medium (DMEM) supplemented with 10% fetal bovine serum (FBS) and 1% penicillin/streptomycin. All cells are cultured in 6-well culture plates under 37°C and 5% CO_2 in an incubator. The medium is replaced every two days, and the cells are harvested and diluted at a ratio of 1:3 twice a week. In experiments, HSCs are pretreated with the experimental concentration of **Oleoylethanolamide (30 μM, 10 μM, 3 μM)** before stimulation with 5 ng/mL TGF-β1. mRNA expression levels of α-SMA (A) and Col1a (B) are analyzed by real-time PCR[1]. **Animal Administration:** [1]Mice[1]
The Sv/129 mice and PPAR-α knockout mice are maintained in a room with controlled temperature (21-23°C), humidity (55-60%) and lighting (12 h light/dark cycles) and given water ad libitum. Mice are randomly divided for methionine choline-deficient (MCD) and thioacetamide (TAA) experiments. In the MCD-diet feeding experiment, wild-type Sv/129 mice and PPAR-α knockout mice are each divided into three groups (n=8 /group): (i) control group receive normal diet; (ii) fed with MCD diet and injected with the vehicle (5% Tween-80+5% PEG400+90% saline, 5 mL/kg/day, 8 weeks, intraperitoneal injection, i.p.); (iii) fed with MCD diet along with **Oleoylethanolamide** administration (**5 mg/kg/day**; 8 weeks, **i.p.**). In another set of experiment, all the wild-type mice and PPAR-α knockout mice are given standard chow diet, and are randomly separated into three groups: the control group is not administrated

TAA or Oleoylethanolamide but is injected with the saline; the TAA group is injected with TAA (160 mg/kg, three times per week, 6 weeks, dissolved in saline, i.p.) plus the corresponding vehicle; the Oleoylethanolamide group is both injected with TAA and Oleoylethanolamide (5 mg/kg/day; 6 weeks, i.p.)[1].

References:

[1]. Chen L, et al. Oleoylethanolamide, an endogenous PPAR-α ligand, attenuates liver fibrosis targeting hepatic stellate cells. Oncotarget. 2015 Dec 15;6(40):42530-40

CAIndexNames:

9-Octadecenamide, N-(2-hydroxyethyl)-, (9Z)-

SMILES:

CCCCCCCC/C=C\CCCCCCCC(NCCO)=O

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA